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THE CLINICAL RECOGNITION AND TREATMENT OF SHOCK

WITH SECTIONS ON "CRUSH SYNDROME" AND "BLAST SYNDROME"

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The recommendations presented in this manual have the approval of the Subcommittee on Shock of the Committee on Shock and Transfusions, and the Committee on Surgery of the Division of Medical Sciences, National Research Council. This information is issued for distribution to civilian doctors, members of the Emergency Medical Service, and civilian hospitals.

FOREWORD

PROMPT ADMINISTRATION of adequate amounts of blood or of a suitable blood derivative (plasma, serum, or serum albumin) is a lifesaving measure in traumatic shock. The development of blood banks and plasma reserves in the United States, therefore, has an important bearing on the treatment of civilian casualties with shock-producing injuries.

With funds made available through the United States Public Health Service, the United States Office of Civilian Defense has given financial and technical assistance to hospitals throughout the United States in the establishment of blood and plasma banks. Some 150 hospitals, chiefly in the target areas, have received grants-in-aid and are preparing plasma to total at least 70,000 units In addition to this reserve, 29,500 units of frozen plasma obtained for the Office of Civilian Defense through the Army and Navy, from blood collected by the American Red Cross, have been distributed. The Medical Division of the Office of Civilian Defense has also procured 50,000 units of dried plasma, from blood collected by the American Red Cross, which are now being distributed. The American Red Cross has placed in its local chapters 5,000 units of plasma which is available to the Emergency Medical Service for treatment of civilian casualties resulting from enemy action. Many hospitals which have not received grants under the Office of Civilian Defense-U. S. Public Health Service program are also preparing plasma reserves which total at least 50,000 units.

Plasma required for the treatment of victims of war-related injuries and major civilian catastrophies is readily obtainable in any community through its Chief of Emergency Medical Service.

Ready availability of plasma, however, is not the final solution of successful shock therapy—this can be attained only when the indications for the use of plasma are promptly recognized and when plasma in adequate amounts is promptly administered.

Various methods of estimating plasma dosage have been evolved to guide physicians in treating shock. Useful as these formulae are, they yield only approximate information. Quantities of plasma calculated by formula frequently do not meet the requirements of an individual patient. It is emphasized, therefore, that in the final analysis the clinical result obtained from plasma therapy is the only reliable guide to adequate dosage. The volume

of plasma necessary to restore the circulation to, or maintain it in, a satisfactory state, and no less, should be administered. Physicians called upon to treat casualties liable to shock must adopt this concept of plasma dosage if deaths from traumatic shock are to be prevented.

CONTENTS

Part I. SHOCK

Part II. THE "CRUSH SYNDROME" THE "BLAST SYNDROME"

THE CLINICAL RECOGNITION AND TREATMENT OF SHOCK

WITH SECTIONS ON "CRUSH SYNDROME" AND "BLAST SYNDROME"

Part I. SHOCK

MODERN WARFARE with its mass bombings has greatly increased the liability of the civilian population to trauma and shock. British experience indicates that 20 to 30 percent of air raid casualties suffer from severe injuries and associated shock. In addition to shock of the classical type following hemorrhage and tissue laceration, two conditions related to shock, but differing enough in clinical aspects to require special consideration, have appeared among civilian air raid casualties. These are the "compression, or crush syndrome" and the "concussion, or blast syndrome."

PRIMARY SHOCK

Primary shock due to neurogenic and psychogenic influences is common among air raid casualties. This syndrome usually follows quickly after the receipt of trauma and unless complicated by blood loss or other shock-producing factors is of short duration. The characteristic circulatory change in primary shock is vasodilation. It is characterized by sweating, relatively warm skin, low blood pressure, feeble and usually slow pulse and syncope. First aid measures that may be of value in early cases include conservation of heat, relief of pain, and the recumbent position. If an early favorable response to therapy is not obtained, or if there is considerable blood loss or damage to the tissues, secondary shock may ensue, unless appropriate anti-

shock measures are promptly instituted. In order that adequate observation and treatment of shock and of potential shock cases may be carried out promptly, special resuscitation facilities should be provided near the ambulance entrance of all casualty receiving hospitals.

SECONDARY SHOCK

A. THE CLINICAL RECOGNITION OF SHOCK AND ITS PATHOLOGIC PHYSIOLOGY

Secondary shock caused by tissue damage is usually later in onset than primary shock and is likely to develop insidiously an hour or more after injury has been sustained. There is no difficulty in the recognition of advanced shock. It is characterized by pallor, weakness, fatigue, thirst, cold perspiration, rapid thready pulse, rapid shallow respiration, low blood pressure, and collapsed veins. The mental state of the patient is not a reliable indication of the severity of shock.

In contrast to the advanced stage, clinical recognition of shock in its early and developmental phases is difficult. Unfortunately there is no pathognomonic early sign. Pallor, tachycardia, and other manifestations which are usually present may be due to other causes. However, the history of trauma, with or without external evidences of injury, in a casualty with a cold pale skin and a pulse of over 100 persistent for several minutes, is presumptive evidence of incipient shock and is sufficient indication for the prompt institution of antishock therapy. Casualties suffering burns which involve more than 10 percent of the body surface area should likewise be considered as potential shock candidates and be treated prophylactically.

The predominant characteristic of secondary shock is diminution in the effective circulating blood volume, accompanied by compensatory vasoconstriction. This fundamental circulatory disturbance results in decreased cardiac output, diminished venous return to the heart, inadequate peripheral circulation, and, finally, impaired blood supply to the tissues. Hemoconcentration and progressive diminution in the blood volume occur. If uncorrected, the peripheral arteriolar constriction which served to maintain blood pressure in the presence of a diminishing blood volume is unable to afford adequate compensation, the peripheral circulation fails, vasodilation ensues, and the blood pressure drops. A fall in blood pressure is therefore a *late* manifestation of shock. Since, due to vasoconstriction, the arterial blood pressure may be normal in the presence of a dangerous reduction in blood volume, *measurement of blood pressure is an inadequate guide to the presence of early shock.* In contrast, blood pressure determinations may be of considerable value in determining the efficacy of

therapy, particularly in regard to plasma dosage. Profound physiologic disturbances which are secondary rather than causative factors are acidosis (diminished alkali reserve), oliguria or anuria, and disturbances of various chemical equilibria.

The initial reduction of blood volume in secondary shock may be due either to loss of whole blood (hemorrhage) or to loss of plasma (burned surfaces, injured tissues), or both. Dehydration, fatigue, pain, fear, and exposure to cold contribute further to its development. During the early phases of shock due to hemorrhage, initial dilution of the blood by fluid drawn into the circulation from the tissues occurs, unless dehydration is present. In inadequately treated shock, especially when hemorrhage has occurred, hemoconcentration due to plasma loss is a relatively late characteristic, representing loss of plasma to the tissues in excess of the loss of whole blood. If adequate therapy is not instituted promptly, such a loss of plasma will lead to irreversible shock much more readily than will an equal loss of whole blood.

When the loss of whole blood predominates over the loss of plasma in a normally hydrated individual, hemoconcentration may not occur even though shock is advanced. In such instances hemoglobin and hematocrit determinations will merely show evidence of anemia.

However, low hemoglobin and hematocrit findings coupled with a low plasma protein level mean acute blood loss, and indicate the need for prompt plasma therapy to combat shock, followed by whole blood transfusion, if the blood loss has been severe. *If deaths from shock are to be prevented, it is necessary to treat prophylactically all patients suffering from injuries which may produce shock, rather than wait until classical signs of shock develop before therapy is instituted.*

B. FACTORS WHICH INFLUENCE THE DEVELOPMENT AND PROGRESSION OF SHOCK

The following sections deal with important points in the management of casualties with particular reference to shock prevention and treatment.

1. Control of Hemorrhage.

Hemostasis should be accomplished by measures which interfere as little as possible with the nutrition of the tissues. For this reason, a tourniquet should not be applied to an injured extremity unless all other means of controlling the bleeding are ineffective. These include elevation of the extremity, the application of a pressure dressing, the application of pressure on the artery between the wound and the heart (use of pressure points), the insertion of a sterile gauze pack, and, if adequate facilities are available, ligation of the vessel. The

method that is used will depend on a number of factors, including the nature of the wound and the facilities that are available for its care.

British experience has indicated that *tourniquets are rarely necessary in first-aid treatment of air raid casualties.* If a tourniquet is necessary, it should be sufficiently tight to interrupt the arterial flow to the peripheral part, since occlusion of the venous return alone results in more harm than occlusion of both the arterial inflow and the venous return. Care should be taken, especially in children, not to make the tourniquet tight enough to injure important nerves. It is desirable to release the tourniquet at least every 15 to 20 minutes. A tourniquet should never be covered with a bandage, and the casualty, if able to carry a message, should be instructed to tell every physician who sees him that a tourniquet is in place. Moreover, the forehead of all such patients should be marked TK with a red skin pencil to indicate that a tourniquet has been applied; its time of application must be recorded on the casualty identification tag which must be affixed to the wrist or ankle of all patients at the incident as soon as possible. After a tourniquet has been in place for some time, complete removal will allow passage of a considerable portion of the circulating blood volume into the dilated vessels of the extremity, and will frequently be followed by the rapid development of shock, unless steps are taken to prevent it. It is wise, therefore, when the tourniquet is removed, to apply a compression bandage to the involved extremity as soon as possible, in order to prevent further loss of plasma from the general circulation into the extremity. If available, the application of an ice-pack to the tourniqueted extremity is desirable.

Improper application or neglect of a tourniquet may result in gangrene and loss of an extremity. If allowed to remain in place too long, but not long enough to produce gangrene, it is possible that a condition analogous to the "compression syndrome" may result. If available, a standard blood pressure cuff provides an excellent tourniquet, but, if not, one may be improvised from a necktie, handkerchief, belt, or triangular bandage.

When a limb is so severely injured that there is obviously no chance for repair, a tourniquet should be applied (and left in place) close to the proximal edge of the wound in order to cut off the whole blood supply to the wounded area and prevent not only loss of blood by hemorrhage, but also loss of plasma into the injured area. In such cases amputation should be performed before the tourniquet is loosened.

When severe shock has already become established, bleeding from wounds, internal as well as external, may have ceased wholly as a result of the associated circulatory changes. Such patients must be carefully observed after resuscitation, since bleeding may begin again

when the blood pressure returns to normal levels coincident with restoration of blood volume.

2. Relief of Pain.

There is evidence that morphine in sufficient dosage is of aid in the prevention of traumatic shock. It should be given for the pain and restlessness associated with injuries, except severe intracranial ones, before the patient is moved from the scene of injury. In air raid casualties, especially when the victim is pinned beneath debris, morphine should be administered as soon as the patient can be reached. Adequate morphine given at this time may do much to lessen the degree of shock attendant upon extrication. While it is possible to administer too much morphine to patients who have sustained injury, ¼ to ½ grain should be given at once to the severely injured and repeated as indicated. In victims subjected to excruciating pain, intravenous morphine may be indicated. The amount of morphine and the time of its administration *must* be recorded on the patient's identification tag.

For restlessness associated with severe intracranial injuries, sodium phenobarbital or sodium amytal may be given in adequate dosage. Morphine in such cases is contraindicated.

3. Immobilization of Fractures.

Splinting of fractures results in a lessening of damage to the soft tissues and blood vessels by sharp bony fragments as well as a reduction of pain. Both factors are important in shock prevention.

Fractures of the long bones should, therefore, be immobilized before the patient is removed from the scene of injury. The most suitable and expeditious way of splinting a fractured extremity in air raid casualties who are buried beneath rubble and debris may be by the use of folded newspapers, pillows, or blankets properly folded and applied. As soon as a casualty can be placed on a stretcher, the use of sandbags may be valuable in further immobilization of the fractured member. Splints should be padded well and bound securely, both above and below the point of fracture, but not directly over it. Fractures of the thigh are particularly difficult to treat by improvised means, but ingenuity and the circumstances surrounding the incident will have to determine, for the most part, the procedure to be followed. Every effort must be made to obtain satisfactory splinting either with a simple splint or a Thomas splint before the casualty is transported.

Experience in England has indicated that the application of traction splints under air raid conditions is usually impossible until the casualty arrives at the hospital. If the casualty must be moved for a long distance over a rough terrain, traction is essential.

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4. Care of Wounds.

The care and promptness of the first aid treatment of open wounds (including burns) is of great importance in determining the likelihood of subsequent severe infection. It is better to leave a wound undressed than to give first aid carelessly.

As a result of British experience with the first aid treatment of air raid casualties, several points may be emphasized. Most air raid victims are extremely dirty and begrimed, their wounds are contaminated with dust, dirt, and debris, making impossible any attempt at adequate cleansing by first aid workers Emphasis should be placed on the rapid removal of casualties from the site of disaster to casualty receiving stations or hospitals, where facilities necessary for proper treatment are available. Unless a wound is bleeding profusely, it should be left alone except for the application of a sterile dressing to prevent further contamination during transportation to a place where aseptic technique can be observed. The chief exception to this rule is in the care of a sucking wound of the chest where immediate closure by any available means must be carried out.

It should be remembered that pathogenic organisms from human sources frequently contaminate wounds after, rather than at the time of injury. Wounds must not be touched by the fingers, unsterile dressings, or other unclean objects. Because of the possible contamination of wounds or burns by droplets from the nose and mouth of the individual giving first aid treatment, adequate masking of the surgeon and the dressing nurse is desirable. It is important to remember that infected and other foreign materials should be removed from wounds by mechanical means rather than by the use of antiseptics.

As soon as adequate facilities are available, scrupulous mechanical cleansing of the wound should be effected. Following debridement, primary closure of wounds may be carried out up to 8 hours following receipt of the injury. Perhaps this time may be extended safely by the local and oral use of sulfonamides. Delayed closure is advisable in wounds of long duration, and those obviously infected. Experience has shown that all hospitals treating casualties should be supplied with sulfanilamide or one of its derivatives, which may be applied locally, or given by mouth or intravenously, as indicated at the time wounds are debrided and dressed.

The prophylactic use of tetanus toxoid or the injection of antitetanic serum is indicated in all air raid casualties with open wounds because of their invariable contamination with dirt.

5. Maintenance of Body Temperature.

Since a definite relationship exists between the onset of shock and loss of body heat, every effort should be made to prevent or correct excessive chilling. If blankets are available, they should be placed

around the patient. More blankets are needed under the body than over it. The injured person should be well blanketed and, during examination and treatment, should be subjected to as little exposure of the body as possible. Preliminary wound dressing should preferably be performed in a warm place. Since heat is lost rapidly through wet clothing, dry garments should be substituted as early as possible. Other measures, such as warming the bed or stretcher on which a shocked patient is to be placed, are desirable. In order to maintain body temperature when the environmental temperature is low, it may be necessary to apply external heat by the use of canteens, hot water bottles, and warm stones or bricks. When these are used, care must be taken that they are placed in such a way as not to produce burns, or excessive warming of the body. Ambulances used for transporting casualties under very cold conditions should be equipped with hot water bottles or other heating devices. Administration of warm drinks is recommended, provided there are no contraindications, such as penetrating wounds of the abdomen.

It must be remembered that excessive heat may exert as severe ill-effects on shocked patients as excessive cold. The extremities are cold in secondary shock, due in large part to the fact that blood is diverted from the skin to other tissues. Too enthusiastic attempts to warm the skin and adjacent tissues by artificial means may produce cutaneous vasodilation and result in further reduction in the circulation to the more easily damaged internal organs. Since dehydration may be present in shock, excessive heating which results in abundant sweating will exaggerate this condition.

It is therefore recommended that patients in shock have adequate cover and be protected against chilling in order to conserve and maintain body heat. Externally applied heat must be employed with great caution; elevation of body temperature above normal is contraindicated.

6. Shock Position.

It is generally accepted that the so-called shock position (head below horizontal) is advantageous, provided it does not increase the patient's discomfort or aggravate his condition. In cases of head injury, however, it is very important that the patient should not be placed with the head below the horizontal level because more hemorrhage from both the scalp wound and brain will occur in that position; in fact, in cases of head injury it is better to have the head elevated. The head-low position, which may facilitate an increased flow of blood to the brain, is desirable in cases in which there is no head injury, because it is well known that nerve tissues are more susceptible to anoxia than other tissues. The venous return to the heart from the larger abdominal vascular areas may also be improved by this procedure, thereby improving the general circulation.

C. TREATMENT OF SHOCK

1. Restoration of Blood Volume.

This is the most important therapeutic measure in the prevention and treatment of secondary shock. The best available method of *permanently increasing a diminished blood volume is by means of the intravenous administration of adequate amounts of whole blood or blood plasma* (*serum or serum albumin*). If there is evidence that the injured patient is dehydrated, crystalloid solutions may be given in amounts not larger than the volume of whole plasma required.

The simplest means of increasing a reduced blood volume in normal individuals, not in shock, is by the oral administration of fluids. Since vomiting frequently accompanies shock and even in its absence fluids are absorbed poorly, other means of fluid administration are usually necessary. The rectal route of fluid administration is relatively unsatisfactory and is suitable only for the introduction of water, since more complicated solutions are inadequately absorbed from the rectal mucosa.

Isotonic salt or glucose solutions may be given by hypodermoclysis. Sites usually chosen for administration are the pectoral region or the thigh, the latter being preferable, unless some local contraindication exists, such as tissue laceration, severe burns, etc. The most frequent indications for the giving of fluid by hypodermoclysis are persistent vomiting or diarrhea.

Since fluid injected subcutaneously is not readily absorbed when blood pressure is low and the circulation retarded, the intravenous route is preferable. It is the most satisfactory one for the immediate introduction of fluids in shock.

(a) *Whole Blood and Blood Derivatives* (*Plasma, Serum, and Serum Albumin*).—The establishment of whole blood banks has eliminated the difficulties encountered in obtaining suitable blood for emergency use in patients suffering from excessive hemorrhage. Blood derivatives (plasma, serum, and serum albumin) are replacing whole blood transfusions in antishock therapy except as a late (nonemergency) measure in instances where there is excessive or continuous blood loss. In addition to the greater ease with which plasma (or serum) can be stored and administered, it has advantages over whole blood in the treatment of shock. Its chief virtue lies in the fact that its protein content restores blood plasma volume physiologically, while at the same time it can be given immediately (no typing or cross-matching necessary) and can be administered much more rapidly than whole blood in whatever quantity may be needed.

Air raid casualties rarely die from hemorrhage. Most persons who die as a result of acute blood loss do so because large amounts of blood

are lost within a few minutes, before medical aid is available. In fact, individuals may remain in relatively good condition with a greatly reduced number of erythrocytes in the circulation, provided an adequate blood volume is maintained. Administration of plasma or serum will restore the vitally needed blood volume, and red cells may be given later if indicated.

Since restoration of a diminished blood volume to normal is the aim to be achieved by the intravenous use of blood or plasma, these substances must be given in sufficient quantity to accomplish this purpose. Various methods have been devised for estimating the amount of plasma or serum necessary for treating a casualty. Though these are helpful, the quantity indicated on the basis of such formulae must be considered only as an approximation. The fact that the calculated amount does not result in improvement does not mean that plasma will not be effective, but usually that additional amounts are needed. Most cases of shock require two to four units of plasma (the equivalent of 1,000 to 2,000 cc. of whole blood), but the final dosage must be based on clinical results rather than on hypothetical calculations.

(b) *Plasma and Serum.*—Plasma is obtained from blood which has been prevented from clotting by the addition of an anticoagulant such as sodium citrate, whereas serum is the fluid part of the blood remaining after the blood has been allowed to clot. The most important constituent of each is protein. The chief difference is that plasma contains fibrinogen, whereas serum does not. The yield of plasma from a given quantity of blood is somewhat greater than that of serum. Other reasons for the more extensive use of plasma than serum are that its preparation is less difficult and it is a natural byproduct of a blood bank.

There is little difference between the relative virtues of plasma and serum, provided both have been properly prepared (i. e., sterile and free from pyrogens).

Plasma is suitable for use without preliminary laboratory typing and compatibility tests and is free from the danger of severe reaction attendant on the use of whole blood. It is not necessary to warm plasma before administration, and it can be given as rapidly as the needs of the recipient warrant. It must be filtered while being administered.

Plasma is now routinely prepared with the addition of a bacteriostatic agent, usually merthiolate (1 to 10,000 final concentration). Phenyl mercuric borate or nitrate in final dilutions of 1 to 15,000 may also be used. (These agents are additional precautions in the preparation of plasma and do not render contaminated plasma fit for use.) Although these preservatives both contain mercury, they are of relatively low toxicity and, in the dilutions in which they are used, are

not dangerous unless very large quantities of plasma are given. Care should be exercised in administering large amounts of plasma containing mercury if there be evidences of renal impairment (see Crush Syndrome).

Plasma is currently stored in one of three ways:

(1) Liquid Plasma.—This is the basic form of plasma from which other types are derived. It has three advantages: 1. It may be stored for 1 year at room temperature without significant alteration, provided a sufficient quantity of 50 percent dextrose is added to make a 5 percent final concentration of dextrose. 2. It can be transported without special precautions, except that it should be kept in its liquid state at a temperature of 55° to 100° F. 3. It is ready for instant use.

(2) Frozen Plasma.—This is liquid plasma quickly frozen at −20° C., usually within 72 hours of bleeding. It is desirable that frozen plasma be kept continuously in the frozen state (between −10° and −20° C.) until required for use, and then liquefied by placing in a water bath at 37° C.

Frozen plasma is somewhat less economical to process than liquid plasma. It requires suitable cold-storage facilities for its preservation, which adds to the expense. On the other hand, it is superior to liquid plasma in that it has a longer period of usefulness (3 years) and because the unstable elements in plasma remain fixed as long as the plasma is frozen. It can be processed either with or without dextrose. The addition of dextrose (50 percent) to make a final concentration of 5 percent dextrose will eliminate almost entirely the precipitation of fibrinogen when the plasma is properly thawed and stored at room temperature. Following thawing, such plasma may be considered usable for 1 year in the liquid state when stored at room temperature.

Plasma may be kept available for immediate use by thawing several bottles of frozen plasma and retaining it as liquid plasma. Frozen plasma may be transported, if properly packaged and kept at suitable temperature by the use of dry ice. Plasma is not altered by repeated freezing and thawing as long as these procedures are properly carried out.

(3) Dried Plasma.—Dried plasma has certain advantages, including satisfactory preservation under much wider temperature ranges and easier transportability. Dried plasma can be transported anywhere; it can be prepared for use, and administration can be begun within a few minutes. It is restored to the liquid state at the time it is to be given by the addition of sterile distilled water containing 0.1 percent citric acid. Complete solution usually results within 2 minutes.

Dried plasma has the additional advantage that it can be reliquefied by adding only enough diluent to make a two to four times concentrated solution: it can then be used to increase the circulating plasma protein in the treatment of hypoproteinemia with or without oedema. Such solutions are of value in the treatment of shock associated with cerebral trauma. Its only disadvantage is its high cost of production.

(c) *Isotonic Salt and Glucose Solutions.*—Although normal saline and 5 percent glucose solutions, administered intravenously, may be indicated at times in the therapy of shock, their value lies chiefly in supplying the needed amounts of water, salt, and sugar. Dehydration may lead to shock and should be treated by the intravenous introduction of solutions of crystalloids, if oral or other means of administration are contraindicated, or do not suffice. Solutions of sodium chloride are indicated when the electrolyte content of the body is reduced. Water containing 5 percent glucose is of value in supplying the body with needed fluid, in addition to the benefit derived from the carbohydrate.

In the treatment of shock, the intravenous injection of isotonic solutions of crystalloids unaccompanied by plasma has, at most, only a temporary beneficial effect, because such fluid soon passes from the circulation (tissue spaces, urine, etc.). The blood pressure and blood volume are often actually diminished after such therapy.

In the presence of gross tissue damage, such as occurs in extensive burns or when large masses of muscle are injured, it is particularly important that large quantities of normal saline or glucose not be given intravenously unless indicated in addition to the administration of plasma proteins. Since protein-containing fluid is lost to the tissues, primary replacement must be made with a similar fluid, such as plasma. Although solutions of crystalloids have a place in the treatment of shock, they should not be used except in conjunction with protein administration (plasma, serum, or whole blood).

(d) *Hypertonic Solutions.*—The occasions are rare in which hypertonic salt or glucose solutions are indicated in shock therapy. While the higher osmotic pressure of a concentrated solution may draw water into the blood stream for a short period of time, the capillary wall is freely permeable to crystalloid substances, and rapid filtration into the extravascular spaces will ensue. When this occurs, protein is lost with the fluid that escapes from the damaged capillaries to the tissues.

The only definite indication for the use of hypertonic solutions is the shock that accompanies severe head injury. If dried plasma is available, the use of two or four times concentrated plasma constitutes a more rational form of therapy. When concentrated plasma is used for this purpose, care must be exercised to avoid excessive tissue dehydration.

2. Surgical Treatment of Casualties Exposed to Shock-Producing Injuries.

(a) *Time of Operation.*—Early operation on casualties is indicated for many reasons. This is particularly true in those instances in which bleeding cannot be stopped by simple means, and in which there are penetrating wounds of hollow viscera, such as the intestinal tract. The length of time separating the injury and operation is an important factor in determining how wounds should be treated, and whether shock and other complications will develop. In general, it may be stated that the mortality will vary directly with the time intervening between the major injury and the operation.

This, however, does not imply that immediate surgery is indicated if the patient is in shock. Before surgery is attempted, shock should be treated by the use of the measures already described. Intelligent management and therapy are required under these circumstances in order that surgery may be done at the optimum time. After a patient previously in shock has received appropriate therapy and has been rendered ready for operation, long and complicated operative procedures should be avoided. The principal lesion should be treated as quickly as the correct type of surgical technique will allow. At the same time, body heat should be preserved, and every effort should be made to prevent further loss of blood and additional tissue damage. *Plasma or blood should be given during and after the operation to prevent relapse into the shock state.*

(b) *Anesthesia.*—Since only in the most exceptional circumstances should any operative interference be undertaken while a patient is in shock, it will rarely be necessary to use anesthesia in shock cases. There are three main points to be borne in mind before administering an anesthetic agent to a patient who has suffered shock: 1. Deep anesthesia in any form is contraindicated. However, it must be remembered that inadequate anesthesia may itself result in shock. 2. The amount of anesthetic agent that will produce light anesthesia in normal patients may be an overdose for such patients. 3. Respiratory function must not be embarrassed by undue stimulation, interference with the airway, or inadequate oxygenation.

The ideal anesthetic to be used in these patients has not been found. Problems which influence the choice of anesthetics are the general condition of the patient, the type of wound, the part of the body involved, and the length of time between injury and operation. In addition to strictly physiologic factors, other considerations are also important in choosing anesthetics for emergency purposes. These include bulk, weight, transportability, explosibility, and fire hazard.

Air raid casualties who are buried under debris require special consideration. They are likely to be in shock, and often suffer from

dehydration. Extrication from wreckage may in some instances be possible only by amputation of a limb. The type of anesthesia administered to such casualties will depend on the part of the anatomy in reach, the choice usually lying between open ether, intravenous morphine, or barbiturates. Patients with facial injuries likewise present special anesthesia problems. Under such emergency conditions intravenous barbiturates may be useful when there is no danger of inhaling blood and when veins are accessible. In some instances endotracheal anesthesia may be necessary.

Nitrous oxide-oxygen anesthesia gained considerable popularity in the last war, due in large part to the observation that animals anesthetized with gas-oxygen mixtures were more resistant to histamine shock than those anesthetized with ether. However, it is now known that surgical anesthesia cannot be produced with nitrous oxide without causing oxygen lack in the brain (anoxic anoxia). Since stagnant anoxia is already present in shock, it is unwise to deprive the tissues of oxygen to any greater degree. For this reason gas-oxygen should not be used as the sole agent in prolonged operations. Nitrous oxide has an advantage in that it is nonexplosive, but its transportation is a serious problem because of the relatively large cylinders required for storage.

Cyclopropane is a satisfactory anesthetic agent for casualties who have suffered from shock, because it can be administered with high concentrations of oxygen and with minimum toxic effects. The greatest drawback to its use is its high explosibility. As with other gaseous anesthetics, there is difficulty in transportation because of cylinder bulk. **Ethylene** presents no significant advantages over cyclopropane.

Ether is probably the safest of all inhalation anesthetic agents, especially when combined with oxygen. It appears likely that its harmful effects, except for prolonged operative procedures, have been overemphasized. It must be remembered that an ether-oxygen mixture is highly explosive and the bulk of the oxygen required for such administration increases the difficulties in transportation. Even without oxygen, ether continues to be one of the most valuable agents, especially if an expert anesthetist is not available. If straight ether is used, care must be taken that cyanosis does not develop.

Spinal anesthesia is ideal for operations on the lower extremities and lower abdomen in patients *in whom the blood volume and blood pressure are not significantly depressed*. The tolerance to hemorrhage is less under spinal anesthesia than under ether. The advantages of spinal anesthesia include ease of administration, ease of recovery, and small bulk of drug necessary. Spinal anesthesia is contraindicated, however, if there is evidence of peripheral circulatory failure, and it should *never* be used on a patient who is in shock or has recently recovered from shock.

Recent advances in the use of **intravenous anesthetic** agents indicate that they may be suitable for brief anesthesia in patients suffering from traumatic injuries. A number of short-acting barbiturates are available and, if given in small amounts repeatedly, they may be of value under special emergency circumstances. The use of these drugs is, however, not without danger. One should avoid large doses of such agents in casualties in poor condition, since they are capable of causing histotoxic anoxia. It is essential that an attendant be present at all times in order to make sure that an open airway is maintained and that a high concentration of oxygen is supplied in the inspired air. Intravenous agents have the advantages of small bulk and weight and freedom from fire and explosion hazards.

Local or regional block anesthesia is the best for operations on patients in shock and should be used whenever possible.

Although not absolutely necessary, it would be wise to have cyclopropane available in casualty receiving hospitals for use especially in cases of thoracic injury. Oxygen should be available for use in conjunction with ether, should the case require it. From the anesthesia viewpoint, all hospitals treating casualties can function quite satisfactorily with a few relatively simple substances; namely, procaine, intravenous barbiturates such as pentothal, and oxygen-ether.

(c) *Penetrating Wounds.*—Penetrating wounds require special consideration because improper treatment of them is likely to result in shock. Certain general principles, to which there may be exceptions, are here outlined regarding the care of such wounds.

All penetrating wounds of the brain are acute surgical emergencies and should be treated by early removal of badly damaged tissue, including devitalized bone fragments and foreign bodies which can be removed without increasing the damage already sustained by the brain. Such wounds should be operated on within 12 hours.

As previously indicated, sucking wounds of the chest should be closed at the earliest possible moment. If injury to the heart and great blood vessels can be excluded, and if foreign material such as dirty clothing has not been carried into the chest by the agent causing the injury, penetrating wounds of the chest should usually be treated by nonoperative means. If the site and nature of the injury suggest damage to the heart, patients should be watched for evidences of cardiac tamponade. If tamponade is present, open operation with suture of the heart wound is required.

Exploration as early as is feasible is indicated in all penetrating abdominal wounds. Perforations of the small intestine, either single or multiple, should be closed transversely to the long axis of the bowel. Resection should rarely be performed, unless there is circulatory impairment. Large bowel perforations should be exteriorized

where possible. If the perforation is sutured, a proximal colostomy should be made. Sulfanilamide powder up to 8 grams should be placed in the abdomen and in the incision during closure. Intestinal distention, which may promote shock, should be prevented or treated by the employment of duodenal suction, intestinal intubation, and inhalations of high concentrations of oxygen.

Bladder wounds should be closed and an indying catheter inserted. If the spleen is badly lacerated, it should be removed. Unless a kidney is severely damaged, it should not be removed.

(d) *Postoperative Care.*—The postoperative care of a shocked patient is similar in many respects to his preoperative care and is as important. *After operation, careful observation and treatment of the patient are necessary to prevent recurring shock.*

Although not absolutely essential, careful laboratory control of the patient who is in shock or has recently recovered therefrom is desirable in order that disturbances in the chemical constituents of the blood as well as hemoconcentration may be detected and, above all, in order that an adequate volume of circulating blood may be maintained. Maintenance or replacement of blood volume by appropriate measures is of primary importance. In the postoperative management of shocked patients there may be further indication for the use of whole blood or plasma in order to correct blood loss from the superimposed injury and trauma of the operation.

Anemia is often present and may require specific therapy. Significant hypoproteinemia may occur in the postoperative period following severe shock, especially if there is associated infection. In the presence of hypoproteinemia care must be taken in administering crystalloid solutions, particularly saline, which may influence the development of or increase in oedema. Hypoproteinemia can be treated parenterally with large doses of plasma, whole blood, amino acids, or casein digest preparations.

D. SUMMARY OF SHOCK TREATMENT

1. Prevent its development.
2. Stop bleeding.
3. Relieve pain.
4. Avoid continued tissue damage by such measures as splinting of fractures, et cetera.
5. Maintain body temperature—prevent chilling—do not overheat.
6. Place in shock position unless contraindicated.
7. Give sufficient whole blood, plasma, or serum as soon as possible.*

*There is no accurate way in which to predict the proper dosage. The important point is to give blood, plasma, or serum early, and to give it in sufficient quantities to produce the desired effects. Any quantity of blood, serum, or plasma less than this means that the patient has been treated inadequately.

8. Administer warm fluids as indicated.

9. Choose an anesthetic agent which will not aggravate the condition.

10. Do necessary surgery as quickly and with as little tissue damage as possible, but only after shock has been controlled.

11. Carefully observe the shocked patient postoperatively. Do not let him relapse into shock. Remember that the primary disturbance in shock is diminution in the effective circulating blood volume. Treat it by intravenous administration of blood, plasma, or serum.

Part II. THE "CRUSH SYNDROME" THE "BLAST SYNDROME"

THE "CRUSH SYNDROME"

Although reports concerning compression injuries followed by peculiar renal lesions appeared in the German literature toward the close of World War I, this syndrome was apparently lost sight of until recently. During the past 2 years a number of clinical and pathological reports, mostly British, have appeared concerning the "compression, or crush syndrome", a condition which results from compression of muscle masses by heavy objects for periods usually of several hours or longer. Most of the reported cases have been victims of air raids who had been pinned beneath debris. Clinically, they present similar characteristic features: The victim is usually in fair to good condition at the time he is extricated from the wreckage and the compression is released; evidences of local injury may or may not be present. Evidences of shock appear within a few to 24 hours; the immediate response to antishock measures is usually good. The general condition of the patient appears satisfactory until some time later (hours to days), when evidences of progressive renal damage develop.

A. ETIOLOGY

Even though the cause of this syndrome is not known, the common denominator is sustained crushing injury to masses of muscle tissue with prolonged ischaemia. There may be an associated loss of nerve function, with anesthesia of the affected part. By some mechanism, at present unknown, damage to the renal tubules results, presumably due either (1) to the local effect of some toxic product resulting directly from the damage of muscle tissue or indirectly as the result of generalized intravascular hemolysis, or (2) to prolonged ischaemia of the kidney tissues, both during the period of trauma and during the period of shock which follows. Since there is often no clinical evidence of shock during the period of compression and there may already be evidence of nitrogen retention before clinical shock appears, the possibility of reflex renal vasospasm as a causative factor cannot be overlooked.

Experimental reproduction of this syndrome by muscle compression has not been entirely successful thus far, in that the renal lesions produced are not histologically the same as those seen in human cases. The other characteristic changes can be produced experimentally, including shock, renal insufficiency, anuria, and typical laboratory findings.

B. PATHOLOGY

1. Muscle.

The damaged muscle shows necrosis due to direct pressure, intrafascial pressure, and oedema, ischaemia, and vascular spasm. There is usually no evidence of vascular thrombosis. In some cases the involved muscle tissue is pale, suggesting loss of muscle pigment (myohemoglobin, actually myoglobin).

2. Kidneys.

The essential lesion is characterized by degenerative changes in the entire nephron, with special damage in the ascending limb of Henle and the distal (second) convoluted tubules. The general arrangement of the renal cortex is preserved. The glomeruli are normal in size. Spherical masses of homogeneous material may be present in Bowman's capsule. Metaplasia of the capsular epithelium near the exit of the tubule is sometimes present. The proximal (first) convoluted tubules universally show degenerative changes with detritus in the lumen. Pigmented casts appear in some instances in the ascending Henle limbs. In the boundary zone of the renal medulla (Henle tubules) are found necrosis and foci of cellular proliferation, sometimes associated with actual rupture and extension of casts into the interstitial tissues. These foci at times involve the walls of vessels, resulting in local thrombus formation. This type of lesion is found most often in patients surviving over 5 days. The distal (second) convoluted tubules are often dilated and show severe degenerative nuclear changes in the tubular epithelium. Pigmented casts are usually present when the patient survives longer than 2 days. Invasion of the tubules with leucocytes is marked in some cases. The more advanced alterations include collapse and partial disappearance of these tubules with early fibrotic changes in the interstitial tissues in the areas which they previously occupied. In the medulla the collecting tubules often contain orange-brown pigmented casts and may show marked evidence of epithelial proliferation.

Attention is drawn to the similarity of this picture to that occurring in persons who die from excessive intravascular hemolysis such as follows incompatible blood transfusion or blackwater fever. In the crush syndrome, however, the pigment has been demonstrated to be myohemoglobin. There is, moreover, insufficient evidence to account for anuria by mechanical blockage of the tubules with pigmented casts.

The urine in the crush syndrome characteristically shows low urea concentration and little evidence of chloride resorption. Mechanical obstruction of part of the tubules would not produce this result, because those which remained unblocked would secrete normal urine. The abnormalities of the urine are such as to support the interpretation that the histologic changes indicate an aberration of tubular function. Failure to concentrate urea and absorb chlorides clearly indicates tubular damage to the kidneys.

C. CLINICAL FEATURES

There is a history as well as local evidence of a compressing or crushing injury to muscle masses for several hours or longer. After extrication from debris and usually on admission to a hospital, the casualty appears to be in good condition. Unless prevented by the application of a tourniquet or pressure dressings before or immediately after extrication, the affected limb rapidly becomes oedematous, with blister formation. Local anesthesia and whealing of the skin are frequently present. The hemoglobin and hematocrit reading are elevated, and unless appropriate antishock measures are instituted characteristic secondary shock develops within a few hours. This usually responds satisfactorily to treatment. The injured limb usually shows diminished arterial pulsation distally and may show evidences of incipient gangrene within a few hours.

Even though the response to antishock treatment appears to be successful, there is progressive diminution in the output of urine, which at this time may show albumin, erythrocytes, and pigmented casts. Progressive nitrogen retention develops, and the patient may become alternately drowsy and restless. Slight generalized oedema, excessive thirst, and vomiting may develop. The blood pressure during this phase of the illness is usually moderately elevated. In the fatal cases anuria finally develops, clear-cut uremia supervenes, and death often occurs suddenly, usually within 7 to 10 days after the injury.

In about 50 percent of the cases, the urinary output can be restored (sometimes spontaneous diuresis occurs), azotemia disappears, the urine clears, and recovery occurs. In general, the patients who survive have relatively less muscle damage. The duration of the compression is apparently not of primary importance in prognosis. Cases have been reported in which the patient survived after sustaining crushing for as long as 26 hours, and deaths have occurred when crushing was present for not longer than 3 to 4 hours.

D. LABORATORY FINDINGS

1. During the phase of shock the usual laboratory findings associated with shock are present, particularly evidences of decreased

blood volume and hemoconcentration. The changes in blood chemistry characteristic of shock have been described. In some cases there is also evidence of fairly severe nitrogen retention at this time.

2. During the period of progressive renal insufficiency there is a diminishing output of urine which is highly acid (pH 4.5 to 6.0) and contains albumin, pigmented casts, and a pigment which has been demonstrated to be myohemoglobin. Microscopic and gross blood may appear in the urine. The concentration of urea is low and that of chloride and creatin high. The blood, and nonprotein nitrogen, potassium, and creatin are elevated. The creatinuria and myohemoglobinuria indicate extensive muscle damage. The hemoglobin and hematocrit fall progressively, and the plasma proteins decrease as nitrogen retention mounts.

E. DIAGNOSIS

The crush syndrome has such a characteristic history and clinical course that no difficulty in diagnosis should be anticipated once the condition becomes established. It is more important, however, to pick out the cases in which the condition may develop and to institute appropriate therapy for the prevention of renal complications. Of special importance will be the history of being pinned under debris. In the absence of history, evidences of crushing trauma should be noted. These may be slight and vary from focal reddening or whealing of the skin to extensive laceration and necrosis.

F. TREATMENT

1. First Aid.

It should be impressed on rescue workers that the real danger to people who have sustained crushing injuries is renal failure. Great care must be exercised in extricating the victim of crush injury. It may be advisable to apply a pressure bandage or possibly a tourniquet to the crushed extremity at the time of release to prevent (1) loss of fluid into the damaged tissues, and (2) passage of absorbed products of muscle breakdown into the general circulation. Steps should be taken as soon as possible to supply abundant fluids by mouth in the form of warm drinks. Alkalies should be given by mouth in sufficient quantity to produce an alkaline urine. On the basis of experimental evidence, immediate application of cold to the injured extremity may be desirable, especially if a tourniquet has been applied. If a crushing injury has occurred, it should be recorded on the patient's identification tag.

2. Hospital Treatment.

On arrival in a casualty receiving hospital, the pressure bandage or tourniquet may be replaced with a sphygmomanometer cuff to allow for gradual release of pressure. This serves to avoid a sudden flood-

ing of the circulation with "toxic" products from tissue necrosis, as well as to prevent rapid loss of plasma into the damaged tissues. It may be desirable, as recommended by Patey and Robertson,* to apply intermittent positive pressure to the affected part.

To prevent the development of shock, all patients who have suffered crushing injuries of the type described should receive prophylactic injections of 500–1,000 cc. of plasma as soon as possible. Large amounts of plasma containing merthiolate or other mercurial preservatives are to be avoided. Fresh liquid plasma, without a preservative, is preferable in treating casualties with crushing injuries. The patient's condition should be carefully watched by both clinical and laboratory observations, and, in the event that evidences of shock develop, more vigorous antishock measures are to be instituted.

Every effort should be made to maintain a satisfactory output of urine (over 1,500 cc. per day). Fluids by mouth and parenterally should be liberally administered, provided steps are taken to prevent the loss of fluid to the damaged tissues. Unless some specific indication for its use is present (low blood chlorides), it is desirable to withhold the administration of parenteral saline solution because of its oedema-producing properties and its possible harmful effects on the damaged kidneys. In its place 5-percent glucose in sterile water is preferable for parenteral administration.

(a) *Diuretics* have been given but are ineffectual; in view of the pathologic changes in the kidneys, little can be expected from them. Certainly the mercurial diuretics are contraindicated.

(b) The administration of *alkalies* to maintain an alkaline urine and presumably prevent the precipitation of myohemoglobin in the renal tubules has been recommended. It is a rational form of therapy although its clinical use has not resulted in striking benefits.

(c) *Amputation* of the involved extremity has been performed in a few cases, but even when accomplished within 12 hours of the injury, has not prevented the development of renal insufficiency.

(d) *Decapsulation of the kidneys* has been recommended, but there is insufficient evidence as to efficacy of this drastic procedure.

(e) *Multiple incision* into the tense oedematous traumatized muscle masses has been carried out in some cases but without striking benefit to the kidney complications. Limb decompression may be indicated as a means of saving the limb when peripheral arterial pulsation has disappeared as a result of excessive oedema, but it seems to have no influence upon the re-establishment of renal function.

(f) *Adrenal cortical substances* have been recommended, in part because of the accompanying shock and also because of the increased blood potassium in crush injuries. The data are insufficient to allow

*Patey, D. H., and Robertson, J. D.; Brit. M. J., Aug. 22, 1942, p. 212.

for conclusions as to their efficacy, and in the reported cases no particular benefit has resulted from their use.

(g) *Atropine and papaverine* have been suggested for the relief of local and renal arterial spasm, but there has not been adequate clinical trial to determine their value. If papaverine is used, it should be given in adequate doses (such as those recommended in the treatment of severe anginal pain). It can be given safely in doses of 1½ to 3 grains three to four times daily, either by mouth or intravenously, depending on the urgency of the circumstances. It is desirable that at least the first dose be given intravenously. The combined administration of papaverine and atropine may be desirable.

G. SUBSEQUENT CARE

If death does not occur and if the injured extremity has not been amputated, the "crush" patient is left with severe muscle damage. Permanent paralysis may result or in some cases shortening with production of Volkmann's contracture may occur. In such instances, the affected extremity should be splinted and physiotheraphy administered as indicated.

H. PREVENTION OF RENAL COMPLICATIONS

The satisfactory management of crush injuries appears to depend primarily on the prevention of renal complications. Prompt removal of casualties from debris, administration of adequate fluids and alkalies, prevention of shock, and pressure therapy to the damaged extremity should all be energetically carried out in every case of crush injury. How effective these measures will prove to be cannot yet be predicted. Prognosis seems to be correlated with the extent of muscle damage.

From the evidence at hand it appears that the ischaemia and anoxia which result from crush injuries are more important in the production of the crush syndrome than the actual damage to the tissues.

THE "BLAST SYNDROME"

Bombing of civilian populations has subjected them to injuries infrequently recognized previously either in civilian or military practice. Although Hooker in 1924 demonstrated that pulmonary tissue was particularly susceptible to blast injury, little attention was paid to this syndrome until the indiscriminate bombing of British cities in 1940. Since then, numerous investigations on the experimental pro-

duction of blast both in air and water, as well as clinical observations on blast casualties, have been reported in the literature. Although air blast injuries are much more likely to be encountered in civilian practice, the essential features of water (immersion) blast are included for the information of civilian physicians should they be called upon to treat casualties suffering from water blast injuries.

A. PATHOLOGY—THE MECHANISM OF BLAST INJURY

Whether due to air or water (immersion) blast, this syndrome is associated with severe shock, and autopsy reveals varying degrees of trauma to certain organs. The central nervous system and the intra-abdominal viscera may be affected, but lung damage is universally present unless the victim's chest was specially protected at the time of the injury.

Because of the relative lack of pathologic studies on human material and in order to determine the mechanism of their production, blast lesions have been reproduced in experimental animals. In some instances, exposure of experimental animals to air blasts will result in death (depending on the severity of the blast and the distance from it) with "bruising" of the lung surfaces and varying degrees of hemorrhagic injury to the lung parenchyma, even though external evidences of injury are lacking. In air blast the shape and extent of the hemorrhagic areas conform roughly to that of the overlying ribs and lateral vertebral borders, suggesting that these structures cause more damage on impact against the lung tissue than do the softer intercostal tissues. In experimental water blast injury to the lungs, it appears that greater damage is produced to the lungs by the intercostal tissues than by the ribs. This discrepancy in observation can be clarified only by further study.

The predominant lesions in the lung are bilateral and are due to hemorrhage from capillary bleeding following alveolar damage and rupture. Associated dilation and congestion of the pulmonary capillaries as well as tiny hemorrhagic areas about the smaller bronchi may also be present. Microscopically the lesser degrees of lung damage appear as areas in which the interalveolar septa, the alveoli, and smallest bronchioles are filled with blood. In locations of more extensive injury, large portions of lung tissue are disrupted. Vesicular emphysema is common in all types of lung blast injury; interstitial emphysema is usually a manifestation of severe blast injury. Although blood may be present in the larger bronchi, free access of blood from the alveoli to the bronchi is prevented in many instances by associated bronchiolar constriction. Occasionally death may be due to asphyxia resulting from occlusion of large bronchi by blood clots. Depending on survival time, pulmonary oedema is found in

varying degrees. Small hemorrhages have occasionally been seen in both the epicardium and pericardium of animals killed by blast. Hemorrhage of the eyes (both anterior and posterior chambers) has been described.

By changing the position of the animal in relation to the blast and by covering portions of the body with protective material (such as sponge rubber), it has been demonstrated that the portion of the body unprotected or facing the blast sustains the greatest damage. Experimental blast injury is, therefore, thought to be due to the direct impact of the pressure component of the blast wave on the body wall. There is no evidence to indicate that pressure changes in the lungs transmitted through the trachea play any part in the production of lung lesions. The suction component of the blast wave appears to have little or no effect in the production of the syndrome.

In water or immersion blast another factor is introduced. Since the human body has roughly the same density as water, the pressure wave is transmitted through the body without displacement, as though the body were water. When the transmitted wave encounters an air cavity in the body such as the lungs, the pressure wave changes into a wave of kinetic energy in the tissues lining the cavity, with disruptive effects. The presence of air in tissues and viscera, therefore, appears to be an important factor in determining their susceptibility to blast injury, especially in water blast, and appears to account in large part for the distribution of pure blast lesions.

Postmortem observations on human casualties dying from the concussion, or blast, syndrome show changes in the lungs similar to those found in experimental animals.

The factors which determine the extent and location of blast lesions are:

1. Size of exploding bomb.
2. Proximity to blast, especially in a confined space.
3. Position of body in relation to the blast wave.
4. Protection afforded by buildings, walls, etc.
5. Amount of local protection.
6. Medium through which blast wave is transmitted.

In order for a man to be killed or seriously injured by the effects of air blast, he must be close to the explosion (within 20 to 30 feet of a 50-kilo bomb) and *unprotected*. With heavier bombs, the danger zone is larger, but to sustain blast injury from even the largest bomb now used, one must be within 150 to 200 feet of the explosion. Because blast waves are more readily transmitted by water than by air, other factors being equal, severe effects will be noted at greater distances in water.

The possibility of a casualty being injured by blast alone is obviously remote. Since, however, several types of injury often co-

exist, primary blast injury should always be considered a possibility in the treatment of a bomb casualty, especially if shock is present out of proportion to the extent of external injury. Trauma due to the impact of the victim against the ground or a solid structure as a result of blast should not be confused with blast injury as here discussed, yet both may be present. This makes it important for rescue workers to note on the patient's identification tag pertinent points concerning location of the victim in relation to bomb craters, protection afforded by buildings, etc. All casualties who have been exposed to blast must be treated as potential blast casualties even in the absence of evidence of external injury, especially if shock is present.

B. CLINICAL FEATURES

The essential difference between air and water blast injuries is in the distribution of the lesions. Water blast is more likely to produce severe abdominal injury, whereas air blast is more likely to produce severe lung lesions. Severe lung and abdominal lesions may, however, occur with both types of blast injury. When blast injury occurs due to either water or air blast, the lungs are always involved unless protected.

1. Pulmonary Lesions.

Casualties suffering from pulmonary damage following blast may be divided into three groups.

Group I. Those dying immediately following the explosion.

In these cases there may be no outward evidence of bodily injury. In some instances blood may be apparent in the nostrils or mouth. At autopsy, the lungs show varying degrees of damage, although in some cases it apparently is not extensive enough to account for death. There may be associated intra-abdominal lesions and central nervous system involvement. Death in many instances is thought to be due primarily to damage to the central nervous system, even though extensive lung damage may be present.

Group II. Those who suffer serious damage to the lungs, not evident immediately following exposure to blast but in whom it is obvious that internal injury has been sustained.

Patients exposed to blast are often thought to be suffering only from shock, but associated lung injury must be suspected. If possible, confirmation of lung damage should be sought by X-ray examination, which is much more reliable than physical signs. Physical signs are often minimal or absent (in the absence of pulmonary oedema) and when present bear no definite relationship to the severity and extent of the lung lesions, usually indicating less lung involvement than actually exists. The patient's general condition is often

a better index of the severity of the damage to his lungs than physical signs in the chest.

Chest pain when present may be of two types—either pleuritic with exacerbation on respiratory movement, or deep chest pain which is constant. Respiratory movement may be restricted in part (1) because of pain or (2) due to the intrathoracic changes which result in emphysema of the lower portions of the lungs with bulging of the lower chest wall. This latter finding is said to be characteristic of certain cases of blast injury. The chest is usually held about three-fourths expanded. Patients may also complain of tightness in the chest or inability to draw a deep breath. Dyspnoea, cyanosis, and tachypnoea are variable symptoms but are usually present in severe cases. Hemoptysis may be present. Cough is usually absent during the first 24 hours following injury. Extreme restlessness is a common symptom and may indicate intracranial damage. Pain in the ears is a frequent complaint and is often associated with ruptured ear drums.

In these patients death often results promptly from shock. If improvement from shock occurs, further intrapulmonary hemorrhage may develop. Pulmonary oedema is frequently present and, if uncorrected, failure of the pulmonary circulation with dilatation of the right side of the heart and evidence of peripheral venous congestion ensue and appear to cause death. Both shock and pulmonary oedema are likely to recur. If recovery from such immediate complications occurs, the clinical course of these cases is similar to that described under Group III.

Group III. Individuals exposed to blast injury in whom there is no evidence of external injury nor suspicion of internal injury at the time of the incident.

The immediate general condition of such patients is good. After varying intervals (usually within 24 hours) cough may be the first indication that there has been pulmonary damage. Chest pain, dyspnoea, cyanosis, and hemoptysis may occur but are not constant. Temperature rise when it occurs early in the course of the disease is attributable to extravasated blood in the lungs rather than to infection. As the condition progresses, temperature, pulse, and respiratory rates increase for several days, then slowly decline as clinical improvement occurs. There is usually a rough correlation between the severity of symptoms and the extent of lung damage. Although shock and pulmonary oedema are infrequently seen in Group III cases, they should be watched for because they may suddenly appear.

Physical signs are variable. Crepitant râles are usually the first physical sign in the chest, followed by areas of tubular breathing and impaired percussion note. Should pulmonary oedema supervene, moist râles appear, their extent depending on the severity and

extent of the pulmonary oedema. If this progresses, increasing respiratory distress and general systemic venous congestion occur. Blood pressure determinations are helpful in assessing the status of the circulation, but are not of value in diagnosing blast injury.

(a) *Differential Diagnosis.*—The history of exposure to blast and the presence of shock out of proportion to the degree of apparent physical injury, with or without pulmonary symptoms, should suggest the possibility of blast injury and lead to observation and X-ray study of the chest. X-ray photographs of the chest show lung lesions with a characteristic mottled woolly appearance. The lack of sputum or scant bloody sputum without significant bacteriologic findings in the presence of pulmonary lesions may aid in making the diagnosis. Bulging of the lower chest wall with poor respiratory excursion is presumptive evidence of blast injury. The status of the ear drums is an important diagnostic point, since pain in the ears and rupture of the ear drums are frequently present. Bacterial pneumonia is the chief differential diagnostic problem.

2. Intra-abdominal Lesions.

Following air blast, injury of the intra-abdominal viscera occurs, though less frequently than lung damage. In water blast the reverse is true. Contusions and lacerations of the bowel may occur, with appearance of blood in the stools. Rupture of the bowel, especially the colon, with production of pneumoperitoneum has been described. Damage is more common to the large than to the small intestine, presumably due to its air content. Laceration or rupture of the urinary bladder, though described in clinical reports, has not been reproduced experimentally. Laceration, contusion, and rupture of the liver, spleen, and kidneys with hemoperitoneum have been described, but whether damage to solid organs or those containing fluid, such as the gallbladder or urinary bladder, is a primary blast effect must be seriously doubted in view of recent experimental observations which indicate that the presence of air in organs or tissues is necessary for the production of blast lesions.

Intra-abdominal injuries occur frequently in the upper part of the abdomen and may be associated with severe pulmonary lesions. One should, therefore, suspect intra-abdominal complications, particularly injury to the hollow viscera, when extensive lung lesions occur near the diaphragm, especially in the costophrenic sinuses. Severe shock frequently accompanies such injuries.

3. Central Nervous System Lesions.

The central nervous system is less commonly affected than either lungs or intra-abdominal viscera, and, when injury does occur, it

is almost invariably associated with damage to other organs, particularly the lungs. Experimental evidence suggests that the central nervous system is not directly susceptible to blast injury, but because the brain and spinal cord are in a bony encasement, lesions are thought to be caused by the sharp increase in hydrostatic pressure resultant from violent back pressure through the venous circulation following compression of the thorax and abdomen. The brain lesions may vary in extent from focal petechial hemorrhagic areas, subpial in type, to extensive vascular thrombosis. There may be similar involvement of the spinal cord. Bloody suffusion and even blood clots about the nerve roots are not uncommon in the more severe cases. In some instances, however, in which death has been attributed to cerebral involvement, significant changes in the central nervous system have not been demonstrable at autopsy.

C. COMPLICATIONS

Late pulmonary complications arise from secondary infection of the damaged areas in the lungs, resulting in pneumonia, pulmonary abscess, or pulmonary gangrene. Intra-abdominal complications, particularly peritonitis, may result from damage to intra-abdominal viscera; the outcome will depend largely on the extent of damage and the success in surgical management of the original traumatic lesions.

D. PREVENTION OF BLAST INJURY

Prevention of blast injury depends on protection of the body from the blast waves of detonated high explosive charges. This may be accomplished by: (1) Protection of the entire body from blast. During air raids, shelter should be sought in protected structures, such as air raid shelters or bomb resistant buildings, or cover should be taken in some sheltered place, such as a ditch, hole, or gutter in the ground, or behind a heavy wall. If out of doors and no physical protection is possible, one should lie on the ground in the prone position, since the back of the chest is less likely to suffer from blast injury than the front of the chest. If in water, one should get as far away from the blast as possible and should avoid partial or complete immersion. If possible, one should stand on a raft or other suitable object which will provide adequate support. (2) Local protection. Although appropriate abdominal and chest coverings made of sponge rubber, kapok, or other shock-absorbing material may partly absorb the blast wave and lessen direct serious lung or intra-abdominal injury, their use for civilians is generally impractical. Such material, however, if kept dry, provides satisfactory protection against both air and water blast.

E. TREATMENT OF INDIVIDUALS EXPOSED TO BLAST.

Persons who have been near exploding bombs, even if apparently uninjured, require observation for at least 24 hours, since the appearance of symptoms of blast injuries to the lungs may be delayed. Great care must be taken not to pass by cases of blast injury with the mistaken diagnosis of "psychic shock."

Shock and pulmonary oedema require urgent attention. In the same patient these conditions may follow each other closely and require heroic measures to correct them, if recovery is to occur. The need for increasing blood volume in shock and the desirability of venesection in pulmonary oedema indicate the complexity of the problem. In a severe case with rapidly developing pulmonary oedema, venesection may be necessary as a lifesaving measure. Shortly after its completion, shock may supervene, requiring augmentation of blood volume. Intravenous administration of fluids other than plasma is contraindicated. If shock develops, plasma should be administered in either isotonic or concentrated form, but must be administered cautiously and in carefully controlled amounts to avoid further embarrassment of the pulmonary circulation.

Because of the complexity of the problem of treating casualties suffering from blast injuries, the status of the circulation must be known at all times, and upon this information, individualized treatment must be based. In some instances it may be possible to control pulmonary oedema by means of oxygen administered under positive pressure* without resort to venesection. As the state of shock progresses, plasma is lost to the tissues, especially to injured tissues, in increasing amounts, and the improvement of the shock state following administration of plasma may actually have a beneficial effect on the pulmonary oedema.

General care should be essentially symptomatic and should follow the accepted procedures for the treatment of pneumonia—complete rest (patient must not sit up or move about even in bed), sedatives for cough, oxygen for anoxia and respiratory embarrassment, and general nursing care. Steps to prevent exposure to secondary pulmonary infection should be taken by isolation of the patient and masking of attendants. If such infection develops, appropriate sulfonamide therapy based on bacteriologic study is indicated. The prophylactic administration of sulfonamides may be indicated in selected cases, beginning preferably 48 to 72 hours after injury.

The proper therapy for intra-abdominal injury will depend on the nature and extent of the trauma. Since casualties with intra-abdominal injury probably will also have suffered serious lung damage, it must be emphasized that surgery is permissible only when the signs

*Barach, A. L., Martin, J., and Eckman, M.: Ann. Int. Med., 12, 754, Dec. 1938.

of an acute abdominal emergency are so definite that operation is urgently indicated. Since inhalation anesthesia is poorly tolerated and because spinal anesthesia is contraindicated due to its shock-promoting properties, local anesthesia with or without supplemental inhalation or intravenous anesthesia is probably the least dangerous.

No specific recommendations can be given for the treatment of victims suffering from central nervous system involvement. In patients who survive, the general and supportive measures usually employed in the care of cerebrospinal injuries should be carried out so long as they do not interfere with the treatment of the pulmonary complications which are almost certain to be present.

Casualties with blast injuries tolerate all forms of physical activity and transportation poorly and should not be moved unless absolutely necessary.

F. PROGNOSIS

Immediate prognosis depends on the extent of injury. If extensive involvement of the central nervous system has occurred, death is usually instantaneous. If the lungs alone are involved, death may occur soon in shock, or later from failure of the pulmonary circulation. If the mechanical effects of trauma are survived, secondary pulmonary complications (due to infection) may appear and prove fatal. If there has been associated severe intra-abdominal injury, prognosis will depend in large part on the nature and extent of these injuries, their satisfactory surgical treatment, and the patient's ability to survive anesthesia and operation.

Failure to recognize this syndrome and initiate early treatment adversely affects prognosis.

Unnecessary transportation is dangerous and may lead to otherwise avoidable fatalities.

Recovery, when it occurs, is apparently complete.

Part III. APPENDIX

INDICATIONS FOR TRANSFUSION

A. WHOLE BLOOD

1. Replacement of erythrocytes (anemia) (stored or fresh blood).
   Loss from hemorrhage.
   Loss from hemolysis.
   Failure of erythropoiesis.
2. Treatment of carbon monoxide poisoning (stored or fresh blood).
3. Treatment of hemorrhagic diatheses (fresh blood).

B. PLASMA OR SERUM

1. Restoration of blood volume in shock (whole or dilute plasma).
   Traumatic shock.
   Surgical shock.
   Burn shock (severe burns require such great plasma replacement that they should be treated with whole plasma rather than with a diluted preparation.)
2. Replacement of deficient plasma protein (whole or concentrated plasma).
3. Supportive treatment of infections and malnutrition.
4. Supplementing prothrombin or complement (fresh liquid, frozen or dried plasma).
5. Convalescent or immune plasma for specific therapy.

C. COMMENTS

Indication No. 1 under "A" is almost never an emergency. When any of the indications under "B" are accompanied by anemia, whole blood may be preferable to plasma. When shock is present, anemia usually need not be treated until the use of plasma has resulted in restoration of the diminished blood volume.

Since the administration of intravenous fluids in patients with heart disease may initiate or aggravate cardiac decompensation, good clinical judgment and extreme care in the administration of blood and plasma are of paramount importance.

DIRECTIONS FOR PLASMA ADMINISTRATION

A. USE AND CARE OF INTRAVENOUS SET

1. The intravenous set illustrated (see fig. 1) is the type recommended for administration of reconstituted dried plasma, but may be used with any suitable rubber stoppered container for the administration of liquid or "thawed" frozen plasma.

2. Assemble as shown in diagram.

3. Improvise suspension sling with gauze or tape.

4. Keep the filter end of the airway tube above the plasma level; if the cotton becomes wet, remove it.

5. Invert the bottle and allow plasma to fill the administration tube. When it is filled and free from air bubbles, stop the flow of plasma by pinching the tube with the fingers or using a shut-off clamp. It may be necessary to "milk" the plasma into the tube at first in order to start the flow.

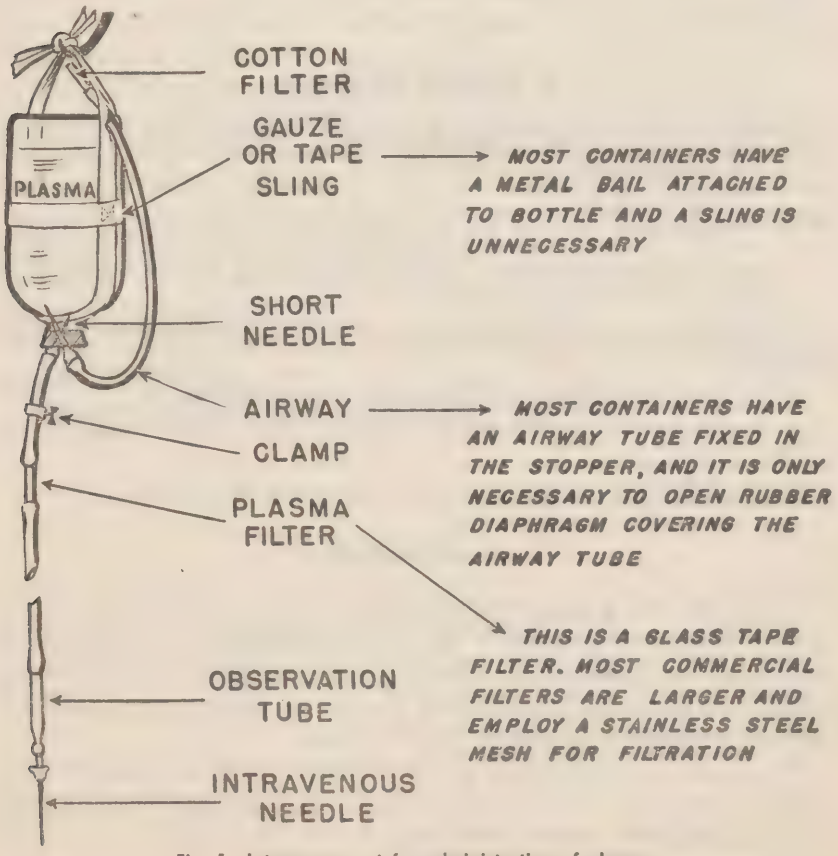

Fig. 1. Intravenous set for administration of plasma.

Fig. 2. Intravenous administration of fluids (including blood and plasma). Note that arm is immobilized by simple splint, loosely applied; needle is held by adhesive tape.

6. Due to the size of the airway needle, the rate of flow of plasma through these sets is rather slow so that a regulating clamp is not normally needed during administration. If the rate of flow is slower than desired, this may be corrected by substituting a long needle, such as an 18-gauge lumbar puncture needle, for the short needle on the airway and adjusting the bottle so that the tip of the long needle is above the fluid level. Temporary increase in the rate of flow may be obtained by applying air pressure through the airway tube.

7. Insert needle in a suitable vein* and fix in place (see fig. 2). If necessary, regulate the flow with a clamp or by changing the height of the bottle. If the patient is to receive additional plasma, shut off the flow of plasma as soon as first bottle is empty but before air enters administration tube. Pull out needles from first bottle and insert in second bottle. Elevate end of airway as before and fix it in place.

8. Administration sets may be reused after cleaning, replacing plasma filters and cotton air filters, and resterlizing.

9. Should an insufficient number of administration sets with filters be available, plasma may be administered by the use of any available type of intravenous burette. The stopper is removed from the plasma bottle and the plasma is poured into the burette, filtering it through 6 or 8 thicknesses of sterile washed gauze. Plasma may also be similarly filtered into a sterile beaker and administered by a sterile syringe and needle.

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*It is recommended that members of shock and resuscitation teams be taught the technique of femoral vein puncture, since in some casualties, particularly those suffering extensive burns, other veins may not be accessible. This technique is easy to learn and extremely valuable when other veins cannot be utilized.

B. PREPARATION OF PLASMA FOR USE

1. Liquid plasma should be kept at a temperature of 55° to 100° F. and is available for immediate use.

2. Frozen plasma must be properly thawed before administration; otherwise, considerable precipitation of fibrin will occur. The precipitation of fibrin does not render plasma toxic but makes it extremely difficult to administer. In order to prevent this, it is necessary to thaw the plasma rapidly in a constant temperature water bath at 98.6° F. (37° C.). Thawing is usually complete within 20 to 30 minutes. The speed of thawing may be increased by providing for constant circulation of water in the water bath. In an emergency, it is possible to thaw plasma adequately in tubs and basins, using the available supply of hot and cold water providing the temperature is maintained with a thermometer at 37° to 40° C. If controlled temperature hydrotherapy baths are available, they will serve ideally for the thawing of frozen plasma.

3. Instructions for reconstituting dried plasma are furnished with each individual package.

4. Plasma or blood should not be artificially heated except by means of a 98.6° F. (37° C.) water bath.

5. Administration of blood or plasma at room or even at ordinary ice box temperature 35° to 42° F. (2° to 6° C.) is not harmful.

Agents Employed in Shock Therapy Which Are of Doubtful Value (Vasoconstrictor Drugs, Oxygen, Adrenal Cortical Extracts) or May Produce Undesirable Effects (Acacia)

1. Vasoconstrictor Drugs.

Numerous vasoconstrictor ("stimulant") drugs have been recommended for the treatment of shock. Included are epinephrine, ephedrine, neosynephrin, coramine, cardiazol, pitressin, benzedrine, paradrine, and paredrinal. Since vasoconstriction is characteristic of secondary shock and vasodilation is not present except as a late manifestation due presumably to anoxia, attempts to induce vasoconstriction by drugs will not be beneficial and may do harm.

In primary or neurogenic shock, which is usually of short duration and often accompanied by vasodilation, the use of vasoconstrictor drugs may be of value. For example, these drugs are of value in correcting the vasodilation and decrease in blood pressure which is often associated with spinal anesthesia.

Although vasoconstrictor drugs may possibly be indicated in the treatment of primary shock, they are not indicated in the treatment of secondary shock.

2. Oxygen.

Although the oxygen content of the arterial blood in shock is usually normal, the tissues suffer from anoxia. This is of the stagnant type, resulting from a decrease in the oxygen content of the venous blood due to the marked diminution in blood flow through the tissues. Although restoration of an adequate circulation is a more desirable means of combatting tissue anoxia than attempts to increase the oxygen content of the arterial blood, inhalation of 95 to 100 percent oxygen may be beneficial both by increasing the oxygen in chemical combination with hemoglobin, and by increasing the amount of oxygen in physical solution in the plasma. Since this provides an increased oxygen pressure in the tissues, oxygen may be of some value in patients with severe shock, especially those who manifest cyanosis of the lips, ears, or nailbeds. Its administration, however, should be considered only when it will not interfere with therapeutic procedures of demonstrated value.

3. Adrenal Cortical Extracts.

The similarity between certain phases of shock and Addison's disease has suggested the use of adrenal cortical substances (adrenal cortical extract and desoxycorticosterone) in the treatment of shock. Clinical proof of the value of such treatment is, however, lacking.

The evidence at hand indicates that adrenal cortical substances alone do not prevent or favorably influence shock. Although these hormones may prove eventually to be of value, they must be considered of questionable value in treatment of shock at the present time. *Their use must under no circumstances be considered substitution therapy for measures which directly replace blood volume.*

4. Acacia.

Six percent gum acacia in 0.9 percent saline was used during World War I in the treatment of shock. Poorly prepared solutions resulted in severe reactions and some fatalities. Although present preparations are of better quality, acacia has gained little, if any, recognition in the treatment of shock. Acacia, being colloidal in character, remains in the blood stream for a longer time than salt or glucose, but in addition to possible immediate reactions resulting from its use it is known to disturb both carbohydrate and protein metabolism for long periods of time, and for these reasons is undesirable. The best opinion, at this time, is that acacia should not be used in the treatment of shock, if whole blood or plasma is available.

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